Seizure 1996; **5**: 147–148

SHORT REPORT

Absence epilepsy in children: the role of EEG in monitoring response to treatment

RICHARD E. APPLETON & MARGARET BEIRNE

The Roald Dahl EEG Unit, The Royal Liverpool Children's Hospital, Alder Hey, Eaton Road, Liverpool L12 2AP, UK

We report the use of repeat electroencephalography (EEG) in the management of 69 patients with childhood-onset absence epilepsy (CAE). Electro-clinical absences were demonstrated in seven children who were felt clinically to have persisting absences. In three of the remaining 62 patients who were thought to be seizure-free, repeat EEG showed electro-clinical absences; revision of AEDs in these three children resulted in clinical and EEG control. EEG is a useful adjunct to the (usual) subjective monitoring of the response to treatment, and should be repeated routinely in all patients with CAE.

Key words: children; epilepsy; absences; EEG; control.

INTRODUCTION

Typical childhood-onset absence epilepsy (CAE) is characterized by sudden, frequent, and brief losses of awareness with or without motor automatisms accompanied by generalized onset symmetrical spike and slow-wave complexes at 3 Hz on the electroencephalograph (EEG)[1,2]. Absences occur spontaneously but are also readily provoked by hyperventilation, a technique used routinely in the EEG laboratory. Such is the sensitivity of this technique in 'diagnosing' this epilepsy syndrome, that many consider that a diagnosis of CAE should not be made if adequately performed hyperventilation fails to induce an electro-clinical attack[2,3]. The response to treatment is usually good with over 70% of patients controlled by a single drug. Monitoring the response to treatment is based largely on report—from the child's parents or teachers (or both), or by hyperventilation in the out-patient clinic. However, these assessments may be misleading for two reasons. Firstly, although reduced in frequency, absences may be missed because of their extremely brief and subtle nature and secondly, hyperventilation may occasionally cause physiological changes ('dizziness' or a glazed expression) which may mimic an absence. As a consequence the child's seizures may be considered (falsely) to be controlled or uncontrolled, respectively. Theoretically, the EEG should be able to provide objective evidence of seizure control because of its sensitivity in demonstrating absences in CAE. However, the role of EEG in monitoring response to treatment in CAE is unclear and has not been formally evaluated other than in patients with poorly controlled absences[4]. We have undertaken a prospective study to assess the value of a 'routine' EEG in monitoring the response to treatment in all patients with CAE.

PATIENTS AND METHOD

From 1 January 1993 to 31 December 1994 all children satisfying the clinical and EEG criteria of CAE were entered into the study. Children with juvenile-onset absence and juvenile myoclonic epilepsy were excluded. The childrens' clinicians were asked to repeat an EEG either as soon as the child's absences were felt to be completely controlled after starting treatment, or, if absences persisted, three to four weeks after initiating therapy. A 'routine' EEG was defined as a recording obtained in the waking state over a

1059–1311/96/020147 + 02 $12.00/0

period of 30 minutes using 16 channels with the surface electrodes applied according to the 10–20 system; hyperventilation was performed for five minutes in both the initial and repeat EEG investigations. It was not the purpose of this study to evaluate either the frequency of sub-clinical epileptiform (spike and slow-wave) activity on the EEG unassociated with any clinical change, or the specific antiepileptic treatment.

RESULTS

In the two-year study period 74 children (59 girls) presented with CAE. The mean age at diagnosis was 5.6 years (range, 3.2–7.9). Repeat EEG was undertaken in 69 patients (93%). In seven patients absences were felt to be persisting, and repeat EEG demonstrated 3 Hz spike and slow-wave activity accompanied by an absence. Sixty-two patients were considered to be fully controlled, i.e. no absences had been witnessed and hyperventilation in clinic was negative. In 59 of these patients (95%), repeat EEG was normal, supporting the clinical impression of seizure control. In the remaining three patients (5%), repeat EEG showed 3 Hz spike and slow-wave activity accompanied by a clinical absence (without any associated motor automatisms); the absences were spontaneous in two patients and induced after three minutes of hyperventilation in one. Following changes in antiepileptic medication, the EEGs normalized in these three patients. No patient experienced any unacceptable side-effects from their medication.

DISCUSSION

This study has supported the hypothesis that absence seizures may remain unrecognized in a small number of children with CAE, and that the 'routine' EEG may be of value in monitoring response to treatment. Whilst we appreciate that a relatively brief, 'routine' EEG may not identify all absences and that a more prolonged period of EEG monitoring[4] (even up to 24 hours), may be more sensitive in detecting a larger number of children with uncontrolled absences, this would not be appropriate or practical in the vast majority of cases (and EEG laboratories). Nevertheless, it would be interesting to compare 'routine', and 24 hour ambulatory EEG in this situation.

The study has not provided any evidence that absences may be falsely diagnosed by the child's clinician in that the EEG remained abnormal with electro-clinical attacks in all seven patients considered to have persisting absences.

It is well-recognised that absence seizures impair awareness and concentration[5] which may, as a consequence, adversely affect memory and therefore learning potential; minimal data have suggested that uncontrolled absences in childhood may contribute to behavioural and cognitive difficulties in adults[6,7]. Therefore, until further confirmatory (or refutatory) evidence becomes available, it would seem reasonable to try and achieve complete seizure-control in CAE, but obviously not at the expense of producing unacceptable side-effects. In our opinion, the EEG facilitates this objective in providing a valuable adjunct in the monitoring of the response to treatment, and therefore in the overall management of patients with this epilepsy syndrome. This issue is not discussed in the current reference books on epilepsy (including paediatric epilepsy) or paediatric neurology. Although our results would suggest that the yield of positive findings is likely to be low, the practice of repeating the EEG is unlikely to result in any significant additional labour or financial burden for most neurophysiology departments.

CONCLUSION

The EEG should be repeated routinely in all children with childhood-onset absence epilepsy as an adjunct in monitoring the response to treatment.

REFERENCES

1. Loiseau, P. Childhood absence epilepsy. In *Epileptic Syndromes in Infancy, Childhood and Adolescence, 2nd ed.* (Eds Roger, J., Bureau, M., Dravet, Ch., Dreifuss, FE., Perret, A., Wolf, P.). London, John Libbey, 1992: pp. 135–150.
2. Aicardi, J. Epilepsies with typical absence seizures. In *Epilepsy in Children 2nd ed.* (Ed. Aicardi, J.) New York, Raven Press, 1994: 94–117.
3. Brett, E.M. Epilepsy and convulsions. In *Paediatric Neurology,* 2nd edn. (Ed. Brett, E.M.). Edinburgh, Churchill-Livingstone, 1991: 342–346.
4. Adams, D.J. and Lueders, H. Hyperventilation and 6-hour EEG recording in evaluation of absence seizures. *Neurology* 1981, **31**: 1175–1177.
5. Browne, T.R., Penry, J.K., Porter, R.J. and Dreifuss, F.E. Responsiveness before, during, and after spike-wave paroxysms. *Neurology* **24**: 659–665.
6. Loiseau, P., Pestre, M., Dartigues, J.F., Commenges, D., Barberger-Gateau, C. and Cohadon, S. Long term prognosis in two forms of childhood epilepsy: typical absence seizures and epilepsy with rolandic (centrotemporal) EEG foci. *Annals of Neurology* 1983, **13**: 642–648.
7. Gastaut, H., Zifkin, B.G., Mariani, E. and Salas-Puig, J. The long-term course of primary generalised epilepsy with persisting absences. *Neurology* 1986; **36:** 1021–1028.